ACCESSION NUMBER: 2002:314562 BIOSIS
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TITLE: Interleukin-1beta expression and phospholipase A2 activation after intestinal **ischemia/reperfusion injury**.
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AB The experiments were carried out to explore the interactions between IL-1 beta gene expression, protein level and phospholipase A2 (PLA2) inhibition after intestinal **ischemia/reperfusion injury** . Using a rat intestinal **ischemia/reperfusion injury** model, after collecting the serum, lung lavage, abdomen cavity lavage and important organ tissue samples from control, injury and PLA2 inhibitor treated groups, IL-1 beta level was measured by radioimmunoassay, and the mRNA expression of IL-1 beta and **type II PLA2** was determined by RT-PCR. After 6 h of injury, the IL-1 beta level in serum was significantly higher than that in the control group; an increase in IL-1 beta was also observed in abdomen cavity lavage 1 or 3 h after injury. IL-1 beta was significantly increased in liver tissue after injury, but was not changed obviously in the lung, kidney and intestinal tissues. IL-1 beta in the lung lavage was significantly higher than that of control group. The mRNA expression of IL-1 beta in lung tissue was increased after injury, but **type II PLA2** mRNA expression was decreased. There were different changes in IL-1 beta level and gene expression after treatment with PLA2 inhibitor chloroquine, cyclo-oxidase inhibitor indomethacin, or PAF receptor antagonist SR27417 respectively after injury. All these results indicate that after intestinal **ischemia/reperfusion injury**, the IL-1 beta level and mRNA gene expression are significantly increased, however, the relationship among IL-1 beta, PLA2 activation and its metabolite release remains to be further elucidated.

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TITLE: Attenuation of ischemia and reperfusion injury of canine livers by inhibition of type II phospholipase A2 with LY329722.
AUTHOR(S): Ogata, Kenji; Jin, Maeng Bong; Taniguchi, Masahiko; Suzuki, Tomomi; Shimamura, Tsuyoshi; Kitagawa, Norihiko; Magata, Shinichiro; Fukai, Moto; Ishikawa, Hiroto; Ono, Takashi; Furukawa, Hiroyuki; Fujita, Miri; Todo, Satoru (1)
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SUMMARY LANGUAGE: English
AB Background. Membrane phospholipid breakdown, caused by ischemia and reperfusion (I/R) of the liver, releases free fatty acids including

arachidonic acids and lysophospholipids, which serve as precursors of various inflammatory lipid derivatives. Phospholipase A2 (PLA2) is a key enzyme that initiates this reaction. In this study, we tested our hypothesis that a **type II PLA2** inhibitor, LY329722, could attenuate hepatic I/R injury caused by a 2-hr total hepatic vascular exclusion (THVE) in dogs. Methods. Eighteen beagle dogs, subjected to a 2-hr THVE, were divided into three groups. Group 1 (n=6) was untreated and served as a control group. LY329722 was administered to animals in group 2 (n=6) intravenously (0.2 mgcntdotkg-1cntdothr-1) for 60 min before ischemia, and to animals in group 3 (n=6) for 60 min starting 15 min before reperfusion (0.2 mgcntdotkg-1cntdothr-1). Animal survival, systemic and splanchnic hemodynamics, hepatic tissue blood flow, liver functions, energy metabolism, hepatic venous thromboxane B2 and endothelin-1 levels, phospholipid levels and tumor necrosis factor-alpha mRNA expression in liver tissue, and histopathologic findings were evaluated. Results. Two-week animal survival was 33% (two of six) in group 1, and 100% (six of six) in groups 2 and 3. LY329722 improved systemic and splanchnic hemodynamics, hepatic tissue blood flow, and energy metabolism, reduced liver enzyme, thromboxane B2, and endothelin-1 release, prevented hepatic phospholipid degradation and tumor necrosis factor-alpha mRNA expression, and lessened histopathologic damage and the number of neutrophil infiltrating into the liver tissue. Conclusion. The present study demonstrated that a **type II PLA2** inhibitor, LY329722, attenuated hepatic I/R injury caused by a 2-hr THVE model in dogs.

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